Management of Cardiac Emergencies*

MODERATOR

HENRY I. RUSSK, M.D., F.C.C.P., Consultant in Cardiovascular Diseases,
U.S. Public Health Service Hospital, Staten Island, New York

PANEL

HARRY GOLD, M.D., Professor of Clinical Pharmacology,
Cornell University, New York, New York

JOHN A. LEWIS, M.D., F.C.C.P., Assistant Professor of Medicine,
University of Western Ontario, London, Ontario, Canada

HUGH E. STEPHENSON, JR., M.D., F.C.C.P., Professor of Surgery,
University of Missouri, Columbia, Missouri

ARTHUR E. STRAUSS, M.D., F.C.C.P., Assistant Professor of Clinical Medicine, Emeritus,
Washington University, St. Louis, Missouri

Dr. Russek: Cardiac arrhythmias, pulmonary edema, acute myocardial infarction and circulatory arrest are conditions which frequently present themselves as emergencies. In most instances, these conditions require prompt attention. Some potentially dangerous conditions are self-terminating and may persist for many days without harm to the patient. On the other hand, there are instances in which the condition is so grave that lifesaving maneuvers may be required instantly. Consequently, the treatment must depend upon the circumstances and must fit the degree of emergency. To begin this discussion of the treatment of some of the major cardiac emergencies, I would like to present a hypothetical case and to ask Dr. Gold the following question: A 45-year-old patient is seen at home following the sudden onset of auricular fibrillation without evidence of congestive heart failure. There is an uncontrolled rapid ventricular rate. There is no history of a similar episode of this nature. I would like to ask what treatment Dr. Gold would recommend and if his therapy would be the same, whether the history and physical examination disclosed a normal heart, a recent acute myocardial infarction, a mitral stenosis, or a thyrotoxic state?

Dr. Gold: Whether I would treat a paroxysm of auricular fibrillation would not be decided by the etiology. If my assumption is correct that the patient you describe, Dr. Russek, had a rapid ventricular rate, but no sign or symptom of circulatory embarrassment, it would be my inclination to have him rest without treatment. The normal rhythm is frequently restored spontaneously.

Dr. Russek: Let's assume now, Dr. Gold, that the patient is apprehensive, that he is experiencing oppression in the chest and that he is moderately dyspneic and yet there is no evidence of frank congestive failure.


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**Dr. Gold:** I would manage the apprehension or panic by psychologic means designed to reassure the patient that he is not facing disaster and if necessary, with the aid of sedation using a small dose of morphine or meperidine (Demerol), or a barbiturate. If I decided the dyspnea here was a symptom of failure, I would proceed with fairly rapid digitalization.

**Dr. Russek:** Now, if in the same hypothetical case the patient were in severe congestive heart failure, your method of treatment then would be rapid digitalization?

**Dr. Gold:** In that case, I would treat him as any severe congestive failure. The regimen would be this: a glass of water followed in an hour by a glass of milk for a total of six glasses of each; 1.2 mg. digitoxin orally followed by a daily maintenance dose of 0.1 or 0.2 mg. as necessary to keep the ventricular rate at about 70/min.; and a daily dose of mercuralluride intramuscularly until the failure is brought under control.

**Dr. Russek:** One final question in this connection, Dr. Gold. Would you have any hesitation in digitalizing a patient who has had a recent myocardial infarction complicated by auricular fibrillation, and if not, do you believe all such patients should be digitalized?

**Dr. Gold:** No, I would have no hesitation in digitalizing a patient with a recent myocardial infarction and auricular fibrillation. As to whether all such patients should be digitalized, the answer is no. Circulatory impairment or very rapid ventricular rate are usually the deciding factors. The dosage is somewhat less than for the patient without myocardial infarction. I take this position on the basis of a laboratory study with ligation of the posterior circumflex artery, which was carried out about 20 years ago in collaboration with President Kennedy's physician, Dr. Janet Travell.

**Dr. Russek:** Would any of the other panelists care to comment in regard to the therapy already described and would any one entertain the use of quinidine in the hypothetical patient under discussion?

**Dr. Strauss:** I would like to comment a bit about this particular hypothetical case which so often becomes a real case. First, as to the use of quinidine, if this is a rapid fibrillator, I think it would be unwise to use quinidine to begin with, although there are many instances where I might use quinidine even without digitalization, but certainly not in this type of case where the heart is beating very rapidly. I would be inclined, in this type case, to try to find what the background is of this individual, if we are speaking not only of the patient with myocardial infarction. It's a different matter if the hypothetical question which you asked just before is about a patient who developed fibrillation from any of several causes. I think one of the things we must always consider in using emergency treatment is first of all, is it an emergency, and second, if it is an emergency, what is the nature of the emergency, to try to make a diagnosis? It does not have to be a complete diagnosis, but at least a definite working diagnosis. I think treating in an emergency manner patients who present themselves without any knowledge of what causes the emergency may often do more harm than good. I would not wait with a rapid fibrillator for the development of even minor signs of cardiac failure, congestive failure. I think that the rapidity of the heart in itself reduces the potential value of the heart as a pump and if we can slow that rapid rate, I think we may prevent some of the things which we want to prevent in patients with myocardial infarction.

**Dr. Russek:** Dr. Lewis, would you favor the use of quinidine in a patient with a normal heart in whom auricular fibrillation has developed in association with a rapid ventricular rate?

**Dr. Lewis:** I would agree with Dr. Strauss. I would use quinidine after the patient has been digitalized, not as a prime therapeutic tool. I would be afraid of increasing the A-V conduction and increas-
ing the ventricular rate. One or two general points I would like to re-emphasize are: first, the importance of determining the etiology; I think that when you see such a patient who is a potential emergency, one must bear in mind long-term management, that is, the emergency treatment for the cardiac patient cannot be entirely divorced from the long-term treatment. We have had bad luck with procaine amide; certainly I wouldn't use it. We have stopped five hearts with it. I would prefer the use of quinidine. This may not be popular with this panel. A person who had rapid auricular fibrillation has potentially serious disease and must be watched. It is quite true that paroxysmal fibrillation is more common than we used to think it was 25 years ago, but I think this man should be regarded as having potentially serious heart disease. I have no hesitation in digitalizing a patient—almost any other patient than a cardiac infarction.

Dr. Russek: Personally, I would like to go on record as favoring quinidine for the patient with a normal heart and paroxysmal auricular fibrillation and also for the patient with mitral stenosis who has developed auricular fibrillation without congestive heart failure. We have found that digitalis glycosides often perpetuate auricular fibrillation in cases of mitral stenosis, whereas quinidine commonly terminates it and with maintenance dosage, may prevent its recurrence. I would like to direct the next question to Dr. Lewis. In a patient with auricular flutter and mitral stenosis with or without congestive heart failure, what would be your recommended regimen of therapy?

Dr. Lewis: I don't think the presence or absence of congestive heart failure will alter the order in which I would use the drugs here. I would digitalize such a patient. If I knew they had had no previous digitalis, I would be inclined to digitalize them rapidly. It is sometimes very difficult to know when a patient has had previous digitalis. Disaster can be induced easily by giving a full therapeutic dose parenterally when in fact the patient is already digitalized. Therefore, I would digitalize him after making sure this flutter is not due to digitalis intoxication. I would digitalize, then give quinidine. That would be my routine of treatment.

Dr. Gold: The patient with auricular flutter usually has an auricular discharge rate of 300 per minute. This is too fast for conduction to the ventricle. Usually, only one-half of the impulses come through so that the ventricular rate is only about 150 per minute, hence a 2:1 A-V block. As the quinidine cumulates, the auricles slow and also the ventricles (or pulse rate), since up to a certain point the 2:1 block persists. For example, the auricles may gradually slow from 300 to 200 a minute with corresponding slowing of the ventricles or pulse from 150 to 100. With more quinidine, the auricles slow further, say to 180 per minute, a speed of impulses which the A-V conducting system can manage. Hence, the A-V block is lifted yielding a 1:1 rhythm. The result is a sudden rise of the pulse rate, for the ventricles now respond to each of the 180 auricular impulses. I presume this is what you meant by the paradox of quinidine action. This is part and parcel of the normal action of quinidine. A sudden rise in pulse may here represent a toxic action, ventricular tachycardia, which sometimes results from the two drugs acting together.

Dr. Russek: Dr. Strauss, I would like to ask this question of you. Would you please detail for us the treatment of ventricular tachycardia in a patient with a recent myocardial infarction?

Dr. Strauss: Well, this really becomes an emergency measure for several reasons. First, ventricular tachycardia is apt to be a very rapid ventricular rate. Secondly, there is always the possibility that ventricular tachycardia may very easily slip over into ventricular fibrillation and that amounts to even more of an emergency, if not death immediately, or practically immediately. I would try to inquire whether or not the patient was hypersensitive, as far as he knew, to quinidine. Most people have taken
quinine at one time or another in their lives. If not hypersensitive to quinine, I then would start in with a relatively small dose of quinidine using the sulfate, giving it by mouth if the patient was not vomiting. I would start in with 3 grains which I would consider my test-dose for idiosyncrasy; two hours later, I would give him 5 grains and in another three or four hours, I would give him 10 grains of quinidine. That happens to be my regimen for using quinidine. I could very easily start him with 5 grains and give 5 grains every three hours until he has taken 30 grains of quinidine or even in dire emergency, 40 grains, although I don’t like to start with that large a dose at first. I certainly would not pay any attention in this type of case to the concentration of quinidine in the blood. I think the important thing is to see what effect it is having on the individual. At the same time, with a rate of that degree, we assume it is a very rapid rate, and in a patient with myocardial infarction he is apt to get a significant fall in blood pressure. I think one of the things we must do in a case of this sort is to use some of the vaso-pressor agents which are available to us in order to try to raise the blood pressure if it has fallen to a significantly hypotensive level. That will do more than just raise the blood pressure. It might actually help to convert the ventricular tachycardia to a normal sinus mechanism. In addition to that, I would feel inclined to use the other measures which one uses in myocardial infarction and that is, give them morphine or other types of narcotics, give them sedation if necessary, place them in a position which is most comfortable under those conditions. I would not be afraid to have him upright even in the presence of fresh infarct and even in the presence of a hypotension, but I would certainly see if the tension or his cerebration would be better in a lower position. I think we have to individualize in this case the same as we must in every person we treat, not only treat according to rote, but treat according to the effect it has upon the individual.

Dr. Gold: I would like to make a remark about the hypotension in tachycardia. Some years ago, a woman walked into my office with a pulse of about 200 per minute. She showed no blood pressure taken by the standard method. I became alarmed, but she seemed quite well; I was quite puzzled for a while when it occurred to me that our indirect sphygomonanometry requires a pulsating antecubital artery and that pulsation might not occur there if the stroke volume is too small because of the rapid heart rate. Over the years, I have seen a number of less dramatic cases of the same sort. They certainly have pressure in their vessels, but we can’t record it by the method we use.

Dr. Strauss: I quite agree with you that the lowering of the blood pressure, especially in tachycardia, but even without tachycardia is not necessarily an indication of shock, and that’s why I emphasized that one has to individualize in each case. I was trying primarily to give a general picture. I would be quite concerned about a patient in whom I could not feel a pulse and if they walked into my office, I would seek further aid. I don’t believe I would let them walk out even though they looked well. One is treating the individual and I think in the usual case which Dr. Russek mentioned use of the vasopressors would be indicated more often than it would not be indicated.

Dr. Gold: In the case of this patient, after I got over my panic and was convinced of the mechanism as well as the fact that she was in no danger, I let her go home.

Dr. Russek: Dr. Strauss, to return to your case of ventricular tachycardia complicating myocardial infarction, would you employ any special precautions in the administration of quinidine in this patient, what is your opinion of pronestyl for such a case, and do you consider digitalis contraindicated?

Dr. Strauss: First of all, I believe I feel very much the same as you in saying that quinidine is an excellent tool. Personally, I
prefer the use of quinidine to procaine amide. As we see medical students and young interns fresh out of medical school, we often find they use the latest rather than the best and I think part of our job as teachers is to try to make them realize that just because a drug or a procedure is new does not mean that it is useless. As far as special precautions with quinidine are concerned, because of the presence of myocardial infarction, I always use precaution in using any of these drugs, be it pronestyl or digitalis or quinidine; I still would use quinidine for its beneficial effects in trying to stop the ventricular tachycardia because I think that is one of the most threatening conditions following a fresh myocardial infarct. Now the other part of your question—is digitalis contraindicated? No, I do not think digitalis is contraindicated. I think that digitalis itself, despite the fact it is said not to, sometimes converts ventricular tachycardia into a sinus mechanism. Of course, in a case of this type, a mechanism may suddenly stop of and by itself with or without medication. Because of that I would not be so sure digitalis is effective.

Dr. Russek: Dr. Stephenson, I would like to ask what you consider to be the best prophylactic and therapeutic measures for cardiac arrest during surgery.

Dr. Stephenson: In spite of the many generalizations about the etiology of cardiac arrest, it is difficult to pinpoint always the most effective prophylactic and therapeutic measures for preventing cardiac arrest in the operating room. The sudden abrupt and complete cessation of all cardiac output from a heart which previously was beating in an effective manner is, as we all know, commonly associated with the vago-vagal response. The role of anoxia, hypoxia and positional changes in potentiating this response are not entirely agreed upon. It does seem, however, that the preventive effect of atropine as related to the problem of cardiac arrest would apply in a large percentage of cases. Atropine is a cholinergic blocking agent which interferes with the action of acetylcholine of the vagus nerves. In spite of impressions to the contrary, a large percentage of sudden cardiac arrests occur in a completely unexpected environment—that is to say, during procedures on individuals who were thought to be in an apparently healthy state. Of the first 1800 cases that we studied, we found approximately one-fourth of all the patients were those in the first decade of life. These are not in a group predominantly with congenital heart lesions, but in relatively normal children. A monitoring of any arrhythmia is essential—particularly to allow one to take preventive measures prior to the onset of ventricular fibrillation. It is for this reason we have urged that all patients, regardless of the operative procedure being performed, be followed by continuous monitoring of the electroencephalogram or electrocardiographic tracing or both. Like the use of automobile seat belts, this preventive measure is not always an easy one to get everyone to adopt. Certain patients are more obvious candidates for cardiac arrest. These would include the anuric patient and the patient with obstructive jaundice.

Dr. Gold: What is your dose of atropine, Dr. Stephenson?

Dr. Stephenson: Well, I think the answer would be ENOUGH. This is not intended to be facetious, but is intended to imply that the dose must be large enough to interfere adequately with the action of acetylcholine on the patterned cell receptor in order to prevent the vago-vagal response. Clinically, a number of investigators have demonstrated that 0.5 mg. (gr. 1/100) intravenously can effectively block the vagal response to distension of the gallbladder, insertion of an endotracheal tube, downward traction on the stomach, and positional changes. It must be remembered that the effect of atropine wears off and that in long operations, it may be necessary to repeat the dosage at intervals of time. If given intramuscularly, the effect probably begins wearing off after about 30 minutes. What would you suggest, Dr. Gold?

Dr. Gold: To block the vagus to the heart in man takes about 2 mg. intraven-
ouslly. That’s a pretty large dose and causes very disturbing side actions.

Dr. Stephenson: To give you an example, we have seen this a number of times, downward traction on the gallbladder will sometimes provoke cardiac asystole. After 0.5 mg. of atropine sulphate given intravenously, the electrocardiographic tracings showed no response to this stimulation.

Dr. Russek: Dr. Lewis would like to revert back to the question of the treatment of ventricular tachycardia. He has some comment he would like to make.

Dr. Lewis: The audience should remember that the panel realizes that the problem of making this diagnosis in the hospital may differ from that of the prospector in the Yukon. The real emergency in this condition is, first, to make the proper diagnosis. If one hears a heart going fast enough to arouse suspicion, Dr. S. A. Levine has taught us two things we should never forget. One is that ventricular tachycardia, if one times it for 60 seconds, shows a slight arrhythmia, as against the auricular tachycardias which have a perfectly regular rhythm. The second maneuver is to compress the carotid sinus. If this produces an alteration in the rhythm, then one can be certain this is not a ventricular tachycardia. Nobody has ever walked into my office with a ventricular tachycardia, but I have seen a good many of these people that appear more dead than alive. This patient may live or die. I don’t take the time to find out whether he is sensitive to quinidine or not. This patient, if he has no blood pressure, and he is obviously shocked, gets 2.5 mg. of metaraminol intravenously at once. If the circulation appears collapsed, as it sometimes has, one injects it intravenously, then extracts blood, reinjects that blood to ensure the metaraminol gets to the central circulation and beyond. Then, if the patient looks as though he is not dying immediately, one gives quinidine intramuscularly. This may be one of the few occasions when in desperation, even without an electrocardiogram, one may have to give procaine amide carefully intravenously.

Dr. Russek: I notice you neglected to mention the third diagnostic finding at the bedside that might be helpful in the identification of ventricular tachycardia, namely, the periodic change in intensity of the heart sounds. Returning to circulatory arrest, there have been a number of reports in the recent literature indicating successful resuscitation following the development of ventricular fibrillation or cardiac asystole in cases of myocardial infarction. Success has been reported with open chest cardiac resuscitation, closed chest compression of the thorax which is being popularized at the present time, application of external electric defibrillation techniques and, of course, the use of the pacemaker in cardiac asystole. I wonder if any of the members of the panel would care to comment on the application of some of these measures in every-day practice.

Dr. Stephenson: In this connection, I might mention a patient that we encountered in the out-patient surgery clinic about a month ago. This gentleman was approximately 65 years old and was being examined for an inguinal hernia by the junior surgical resident. The patient suddenly fell over in a state of collapse, there was no pulse or blood pressure and he appeared to be dead. He was immediately placed on the floor and the now standard method of closed chest compression was begun. It was assumed that ventricular fibrillation was present. This was verified by an electrocardiographic tracing in the next few minutes. Of course, this made little difference relative to his immediate management. Adequate cerebral oxygenation was being accomplished. We had a second case that same day in an 18-year-old girl and were able to use closed chest compression prior to an emergency thoracotomy for pericardial tamponade. Both of these patients died subsequently. There is no question that perfusion of the vital organs can be accomplished by this technique. It is not an easy technique to master and is much like the open
heart approach using intermittent ventricular compression. It does require a knowledgable individual who has had some practice. In the case of the first individual, an external cardiac electrical defibrillator was brought into the room and after the first shock, the heart returned to normal sinus rhythm. He was admitted to the hospital where the electrocardiogram subsequently revealed an anterior myocardial infarct. While the closed chest compression method is a worthwhile adjunct to our armamentarium, I do not think it is any panacea. I think it is still to be shown conclusively that the result can be better in the operating room with closed chest than with the open approach. Some have had good results, but I might point out that in the first two years our hospital at the University of Missouri was open, we had an 80 per cent success rate with the open approach. I think we must feel confident that we can have this same degree of success with the closed chest approach. Unquestionably when the arrest occurs outside the operating room, in the doctor’s office, in the emergency room, etc., it is a worthwhile procedure; it will save lives and should be instituted—particularly in cases of accidental electrocution and drowning.

Dr. Strauss: May I just add one word and that is while making sure the circulation is carried to the vital organs, one must not forget to insufflate the lungs periodically so that they also get air. That’s a very simple thing with mouth-to-mouth inflation.

Dr. Stephenson: Dr. Strauss is entirely correct. The importance of maintaining adequate oxygenation by the mouth-to-mouth method of artificial respiration cannot be over-emphasized. As one of your former pupils at Washington University, Dr. Strauss, I apologize for not having emphasized this earlier in my discussion. All too often the main effort is centered on reinstating a normal cardiac rhythm and the airways are neglected.

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ROENTGEN MANIFESTATIONS IN LUNGS IN MILK ALLERGY

A group of infants shown to have high levels of precipitins against cow’s milk manifest a characteristic syndrome of chronic cough, dyspnea, and wheezing, with recurrent episodes of pneumonia. Frequently other respiratory and gastrointestinal symptoms and iron deficiency anemia are also present. These findings disappear soon after withdrawal of milk products from the diet.

Pulmonary hemosiderosis was found in four of the eight cases discussed. It is intriguing to implicate milk allergy as one possible cause of this idiopathic syndrome. Indeed, one of the primary etiologic theories has been that of a hyperimmune reaction in the alveolar walls to some unknown allergen: milk protein appears to be such an antigen. The authors emphasize the importance of recognizing the possibility of milk allergy as a cause of chronic and recurrent respiratory and gastrointestinal illness in infants who show a nonspecific and variable pattern of pneumonias.


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HEMOPTYSIS CAUSED BY ASPIRATED GLASS FOREIGN BODY

A case of aspirated foreign body with apparent cure following lobectomy has been reported. This case merits attention because the only symptom was hemoptysis, and the offending object, a fragment of glass. Neither of these has been reported previously.